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THE PLANT DISEASE REPORTER

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THE PLANT DISEASE SURVEY

Division of Mycology and Disease Survey
BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING
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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Division of Mycology and Disease Survey serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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THE PLANT DISEASE SURVEY
DIVISION OF MYCOLOGY AND DISEASE SURVEY

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BIG-VEIN OF LETTUCE IN NEW YORK STATE

Charles Chupp and W. C. Paddock

The virus disease big-vein has been found sporadically on Long Island ever since Jagger first reported it in California in 1934, but it never was troublesome enough to cause any concern to the commercial lettuce growers until this spring when the situation reversed itself entirely. The disease is appearing in epidemic form not only on the Island, but specimens were sent from Kingston, Ulster County, where 40,000 plants out of 200,000 were affected.

An inspection trip was made to Nassau County, May 23. Fields were examined about Hempstead, Elmont, Hicksville, Wantagh, and Melville. The losses on individual farms ranged from 10 percent to so nearly total that the fields were plowed under before harvest time.

Apparently an unusual combination of weather conditions was responsible for the sudden appearance of the symptoms. The winter was warm enough so that the soil temperatures remained fairly high, even in February. Then in March and again in April there were brief periods of unseasonably high temperatures accompanied by heavy rains.

Judging from the appearance of the affected plants, there were at least three infection periods and possibly a fourth. Many seedlings were killed, but it could not be determined whether big-vein was responsible for this early injury or not. There probably was one infection period in March or earlier, one a few weeks later, and one in which the leaf symptoms were just beginning to show.

The symptoms mentioned by Jagger and Chandler are those of beginning infection. They mention light yellow leaf veins. The Long Island material was pale yellowish green on Big Boston and Iceberg types but almost white on the Cos variety. There was a band along each side of the vein as well as the vein itself, so that the combined streak appeared large, and with its frequent anastomosing made a pleasing pattern bordering the darker green interveinal areas. As the symptoms progressed, the leaves became slightly thicker and crinkled, with the wrinkles bulging upward and again making the veins appear larger than normal.

The more seriously and earlier-infected plants were much stunted in growth. They produced no heads and either had longer outer leaves and shorter inner ones, or the outer leaves were shorter and the center leaves somewhat developed, resembling a lettuce plant about to send out a seed stalk. When the outer leaves were longer, the symptoms resembled slightly aster yellows on lettuce, but there was no whitish to yellowish rosette of dwarfed leaves in the center, nor were there

white blisters along the margins of the youngest inner foliage.

There was no pronounced mottling of any part of the plant, but beginning with the older outer leaves and slowly progressing upward, the leaves showed advancing stages of yellowing or bleaching of the parts between the veins and finally necrotic stipples, spots, or blotches, as are sometimes present when plants show extreme symptoms of magnesium deficiency. There was some evidence indicating that when infection began early enough and with a great enough severity, the entire plant died, for there were many dead plants that did not show symptoms of Sclerotinia drop or Botrytis gray mold, the two diseases commonly associated with dying lettuce on Long Island.

Little evidence could be obtained regarding the source of the inoculum or the ecological relations that made the symptoms so startlingly evident during the present season. On one farm, however, Big Boston was seeded in February. Half of the field was planted directly, while the plants for the other half were grown until the last of March in cold frames. The transplanted seedlings developed almost normally and produced a commercially profitable crop. The direct seeding, however, showed nearly 100 percent loss. Presumably the cold frame soil was not infested or the temperatures were too high for infection, while that of the field had a heavy inoculum potential, so that the field-grown plants were subjected to the first two periods of heavy infection.

In the Wantagh area much of the lettuce was grown on heavy sticky soil as compared to the light sandy types of most of Long Island. Judging from the appearance of this heavy soil, it retained water tenaciously and no doubt had a much lower temperature than prevailed during March and April in the light sandy areas. Consequently the lettuce crop about Wantagh showed a much lower percentage of visible infection, and this was of a milder or more recent development.

Pryor, in a table showing the effects of soil temperature on symptom expression of big-vein, gave almost as high a percentage of disease at 57° F. as at 72° F. If our brief observations are correct, there is a very sharp drop in symptom expression below 57° F., and during the average year, the soil temperatures during March and early April are too low for big-vein to be destructive. It has been shown during previous seasons that as summer weather with its high temperature approaches, the disease disappears so far as its visible effects on the plants are concerned.

There seemed to be no difference in susceptibility among the three types of lettuce observed, but when the crops were infected, the commercial value was lowered most in Big Boston, somewhat less in Iceberg, and appreciably less in the Cos or Romaine type.

CORNELL UNIVERSITY, ITHACA

A STRAIN OF STREPTOMYCES SCABIES PARASITIZING CAYUGA POTATOES IN IOWA

W. J. Hooker and C. E. Peterson[1]

Variation within Streptomyces scabies (Thaxt.) Waksman & Henrici has been recognized and physiologic races have been reported, although it has usually been observed that a potato variety resistant in one location is resistant elsewhere. Certain exceptions to this generality have been observed within the last few years.

In the fall of 1948 deep (type 4) scab developed on tubers of the Cayuga variety grown at Clear Lake, Iowa. In previous years this variety ranked extremely high in scab resistance in Iowa, exhibiting a few very shallow (type 1) lesions. As far as could be determined, there was no varietal mixture in the seed lot as plants and tubers were generally similar in type. The only unusual characteristic was the deep scab.

Greenhouse inoculations were carried out with eight isolates from three different Cayuga tubers. These isolates were grown on potato-dextrose agar and the spores and aerial mycelium were suspended in water and thoroughly mixed with autoclaved peat soil. Cayuga seed pieces previously disinfected by the hot formaldehyde method were planted in the infested soil.

Scab was relatively severe on the Cayuga tubers grown in soil infested with two of the isolates. Lesions were generally deep and numerous. Tubers were free of scab in the uninoculated controls and with the other six cultures which were apparently nonpathogenic. Comparisons were made with an Iowa isolate of Streptomyces scabies known to cause severe scab on susceptible varieties and type 1 or 2 lesions on resistant varieties. In general, the two isolates obtained from deeply scabbed Cayuga caused appreciably deeper scab on Cayuga than did the culture representative of the usual Iowa isolates.

Further studies are under way to establish the validity of the field observation and the preliminary greenhouse inoculation trial.

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[1] Cooperative investigation between the Division of Fruit and Vegetable Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, and the Iowa Agricultural Experiment Station. Ames, Iowa

BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, AND THE IOWA AGRICULTURAL EXPERIMENT STATION

VASCULAR BROWNING IN DADE COUNTY, FLORIDA GREEN-WRAP TOMATOES

Robert A. Conover

The winter growing season in 1948-1949 in Dade County, Florida, was the most favorable for tomatoes in many years. Vine growth was heavy and exceptional yields of fruit were reported by many growers. However, the proportion of marketable fruit was lessened considerably by a vascular browning occurring in and around the bundles of the fruit wall. This condition, known locally among the growers and tomato buyers as "gray wall," caused an estimated loss of 10 to 20 percent of fruit maturing during late January and February, with losses in some fields ranging as high as 70 percent. The incidence of vascular browning was noticeably less during March, but it was still showing up as late as May 1. This malady was probably first recorded in this area in 1927 by Dr. G. B. Ramsey. His field notes and photographs for that year, which he kindly sent to the writer, record a condition indistinguishable from that herein described. Vascular browning has been seen annually in Dade County since 1937, but this is the first year it caused important loss. It has been noted also in the other major tomato growing districts in Florida but estimates of losses in these areas are unavailable.

It is possible that vascular browning is also known in Texas judging from Young's[1] discussion and illustration (Fig. 29, p. 51) of "internal browning and core rot." Young suggests that internal browning may be one form of core rot, and that both core rot and internal browning may result from the same cause as blossom-end rot. In this connection it is interesting to note that in Dade County neither core rot nor blossom end rot was seen in commercial fields this season.

Vascular browning appears externally on the fruit as a grayish-brown discoloration seen through the somewhat translucent outer wall of the tomato. The margin is usually indistinct and the discolored areas vary from nearly circular to longitudinal sectors in that portion of the fruit wall covering the locules. This discoloration is more noticeable on the sides of the fruit, but has been seen extending from the stem scar to the blossom end. In milder cases, only one or two such areas may be seen on one fruit, in which case the surface of the fruit is smooth. In more severely affected fruit, the entire tomato may be discolored and the skin somewhat corrugated but not broken. In cross section the affected area is dark-reddish brown and is centered in and around the vascular bundles of the fruit wall. In mildly affected fruit only the vascular bundle is discolored; more severely affected fruit show a lateral extension of browned tissue which, in some cases, involves the entire fruit wall. The

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[1] P. A. Young. Tomato Diseases in Texas. Tex. Agr. Exp. Sta. Circ. 113. 1946.

septa and placental tissue are not involved. Fruit of all sizes larger than one-half inch may be affected. If mature, such fruit ripen without further breakdown, and in a fully ripe fruit, the discoloration is usually masked by the red fruit color. Internally, however, the browned areas are still present.

Vascular browning may show up at any picking, although it was more common in the third picking this season. In many cases vines bearing affected fruit subsequently set fruit that matured normally without any vascular browning. The fruit on individual plants show great variation in the amount of vascular browning present. Only one fruit per plant may be affected, or every fruit on successive hands may show this disorder. From one to all the branches of a plant may bear such fruit. While in general, the distribution of affected plants in the field appeared to be at random, in some fields more vascular browning appeared in certain areas than in others. Nothing consistent was attendant to these variations that would aid in explanation of the distribution. All commercial varieties grown in this area, as well as many varieties planted experimentally, developed vascular browning. The condition occurred with all fertilizer practices used here, including the use of copper, zinc, and manganese nutritional sprays. In one case at least, vascular browning appeared in the so-called hydroponic farms where particular attention is paid to keeping a balanced nutrient solution, including minor elements. The only consistency in the whole picture has been that vascular browning is usually more severe where vines are luxuriant and the fruit heavily shaded.

At present no explanation for the cause of this malady is available. Repeated microscopic examination and attempts at isolation have uniformly failed to reveal the presence of an organism. This, combined with the failure of the fruit to break down after picking, indicates that the cause is either physiological or virus in nature. Field observations do not consistently support either hypothesis.

UNIVERSITY OF FLORIDA SUB-TROPICAL EXPERIMENT STATION, HOMESTEAD, FLORIDA

WATERMELON DISEASES IN THE GAINESVILLE AREA OF FLORIDA THIS SEASON

George F. Weber, John H. Owens, and John F. Darby

Anthracnose (Colletotrichum lagenarium). None was observed on plants in the field or on melons at the various railroad car loading points in the Gainesville area during an extended survey of several hundred acres over which the disease has appeared annually. Weather has been dry with above normal temperatures.

Gummy stem blight (Mycosphaerella citrullina) is probably more prevalent on this year's crop than for several years in the past. It is present in almost every field, large or small and regardless of source of seed, being more common and destructive on the Cannon-ball variety than on Tom Watson, however. The foliage lesions have been unusually plentiful. On one 100-acre field 100 percent infection was noted, with 5 percent killed foliage and the first picking possibly two weeks off. Considerable damage to vines was evident with the fungus sporulating plentifully. Considerable loss from the disease is expected.

Fusarium wilt (Fusarium niveum). [F. oxysporum f. niveum] is the most prevalent and destructive disease on melons in this area this season. The severity varies from slight traces in new soil and on disease-resistant varieties, to 100 percent infection and 50 percent loss in plantings of common varieties on old cultivated lands, even though they had "laid out" for a decade or more. The dry (less than an inch of rain) six-week period previous to June 1 undoubtedly was an exceedingly important factor in the present manifestation of the disease. The lack of rain and resulting higher temperatures are doubly responsible for the excessive prevalence and destructiveness of the disease.

The following samples were taken over a 20-mile range:

100 acres	100% infection	50-60% dead plants
20 acres	90% infection	10% dead plants
40 acres	80% infection	15% loss
20 acres	20% infection	2% dead plants
120 acres	10% infection	No loss

Other diseases: Some soil-rot (Corticium vagum) [Rhizoctonia solani]. Probably less than 1 percent was observed on melons, resulting in rejection at the car-loading places. Field examinations showed the disease frequent and evident enough to cause rejection for shipping. Powdery mildew (Erysiphe cichoracearum) was observed but once on a protected margin of a field. Several plants showed southern blight (Corticium rolfsii). Its scarity was noticeable compared with previous years.

Stem-end rot (Diplodia natalensis Evans) was not observed. In examining railroad car loadings it was estimated that probably one car in a hundred was being treated for the transit disease by reclipping stems and painting with a fungicide.

COLLEGE OF AGRICULTURE, UNIVERSITY OF FLORIDA, GAINESVILLE, FLORIDA

WILDFIRE EPIDEMIC IN PLANT BEDS

Howard E. Heggestad

Widespread occurrence of wildfire (Pseudomonas tabaci) was observed in tobacco plant beds of East Tennessee this spring. The disease was first apparent about the middle of April when growers began weeding beds. At this time, leaves on most plants were from 1/2 to 2 inches in length and the plant growth nearly shaded the ground. Diseased plants were conspicuous due to the contrast between the characteristic lemon yellow circular lesions and the succulent green plant growth normally found at that period. Frequent rains favored disease development. Plants in small areas were killed as the bacterial toxin spread to the growing point. Inspection of several beds where premature reports of blue mold were received revealed many cases of wildfire. Severe cold injury was found in a few beds.

Later spread of wildfire in the beds may be attributed to weeding while leaves of plants were wet and to the activity of tobacco flea beetles. Also, a high percentage of the plant beds have the cotton covers lying directly on the plants with the edges fastened to the ground by wires through the grommets rather than fastened to board frames or poles. Early infection is difficult to detect in these beds; flea beetle injury is more prevalent; and, no doubt, considerable plant injury and spread of wildfire occurs when the wet cotton cover is moved for any reason: for example, to pull weeds.

The writer observed on three different farms that growers had wildfire in beds not treated with the recommended bordeaux control treatments but no wildfire present in nearby treated beds. Growers having wildfire either failed to make any treatment or applied the treatments too late. Many untreated beds were completely destroyed by wildfire.

The disease, although present, becomes less conspicuous as burley seedlings are hardened for transplanting. Usually when hardened the top leaves turn yellow and bottom leaves become completely chlorotic. Although many growers do not know they have wildfire, it is estimated that 50 percent of the plant beds in Greene County, and also in other East Tennessee counties, have some wildfire present. Most growers have

transplanted infected plants because they were not aware of the presence of the disease or because healthy plants were not available. This unusual widespread occurrence of wildfire in plant beds is associated with increased prevalence of the disease noted in the field in recent years.

U. S. DEPARTMENT OF AGRICULTURE AND TENNESSEE AGRICULTURAL EXPERIMENT STATION

EFFECT OF BORDEAUX AND PETROLEUM OIL ON THE ERADICATIVE ACTION OF MONOCALCIUM ARSENITE AGAINST SCLEROTINIA LAXA IN APRICOT TREES

E. E. Wilson

In 1942 this writer[1] reported that a spray preparation containing 3 or 4 pounds of monocalcium arsenite per 100 gallons of water was highly effective in suppressing the conidial inoculum of Sclerotinia [Monilinia] laxa in stone-fruit trees. Bordeaux mixture 4-2-100 and 8-4-100 had little effect on a 4-100 preparation but reduced the eradicative action of 1-100 and 2-100 preparations. Recently, however, Calavan and Keitt[2] concluded from tests conducted against S. laxa in Wisconsin that 6-4-100 bordeaux and 3/8 percent menhaden fish oil increased the efficacy of monocalcium arsenite. Three-eights percent Stanolind spray oil, they found, could be substituted for the fish oil without loss of effectiveness.

Table 1 contains results obtained under California conditions in 1949. Since fish oil was not available at the time needed, a heavy emulsifiable petroleum oil preparation (Ortho-Adhesive) was substituted. Bordeaux, 4-4-100, was prepared in the sprayer tank and to this was added 3 pounds per 100 gallons of monocalcium arsenite and 1/2 percent of the petroleum soil. In mid-January, 1949, this preparation and one containing 3 pounds of monocalcium arsenite per 100 gallons only were applied to different blocks of apricot trees bearing numerous brown-rot-blighted twigs. In addition, these blocks received bordeaux, 10-10-100, at the pre-blossom (red-bud) stage.

At the time of giving the eradicant treatment, very few sporodochia of Sclerotinia laxa were present on the blighted twigs. These developed in large numbers, however, during February. Hence, the action exhibited

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1 Wilson, E. E., Experiments with arsenite sprays to eradicate Sclerotinia laxa in stone-fruit trees as a means of controlling the brown rot disease in blossoms. Journ. Agr. Res. 64: 56-594. 1942

2 Calavan, E. C., and G. W. Keitt. Blossom and spur blight (Sclerotinia laxa) on sour cherry. Phytopath. 38: 857-882. 1948

Table 1. Comparison of monocalcium arsenite with and without bordeaux and petroleum oil as eradicant treatment for controlling brown-rot blossom blight in apricot trees.

Orchard No.	Treatment[a], pounds per 100 gallons or percent by volume	Percent reduction in sporodochial development	Percent reduction in twig blight[b]
1	Monocalcium arsenite, 3-100	87	99
	Monocalcium arsenite, 3-100; plus bordeaux, 4-4-100; plus 1/2 percent petroleum oil	80	99
2	Monocalcium arsenite, 3-100	67	80
	Monocalcium arsenite, 3-100; plus bordeaux, 4-4-100; plus 1/2 percent petroleum oil	58	75
3	Monocalcium arsenite, 3-100	91	99

[a] All plots received bordeaux 10-10-100 at the pre-bloom stage.

[b] Compared with an average of 21 percent blossoming twig blight in eight orchards which recieved only bordeaux 10-10-100, at the pre-bloom stage.

by the eradicant spray was primarily one of suppressing sporodochial development rather than that of destroying those present at the time of treatment.

Inasmuch as all apricot trees in the test orchards were given the eradicant treatment, four nearby orchards, which received only bordeaux, 10-10-100, at the pre-blossom stage, were utilized as controls. Those selected for this purpose were similar to the test orchards with respect to the abundance of hold-over blighted twigs. In all, moreover, conidial development ran uniformly high, ranging from an average of 10 to an average of 16 sporodochia per blighted twig. Consequently, it is reasonable to assume that, except for the presence of the eradicant spray, conditions were favorable for abundant sporodochial development at the test orchards as well.

Frequent rains occurring throughout the blossoming stage of the apricot trees produced several periods favorable to blossom infection by

Sclerotinia laxa and interrupted the application of protective sprays. Consequently, variations in the incidence of blossom blighting between orchards or areas of the same orchard seem likely because of differences in the timing of the protective sprays during this critical period. To determine the variability of blossom infection among bordeaux-sprayed trees, therefore, eight orchards (including the four orchards utilized as controls for observation on sporodochial development) in the vicinity of the test orchards were examined for the disease. The trees in these orchards bore numerous hold-over blighted twigs. All orchards were sprayed with bordeaux, 10-10-100, at some time during the period from about March 4 to March 15. The following percentages of blighted blossoming twigs were found: 8, 14, 15, 17, 19, 27, 31, 35 (average 21 percent). Test orchard No. 1 and the third control orchard (that with 15 percent infection) were adjacent and received bordeaux on the same days. For comparing the incidence of infection in orchards receiving bordeaux only with that in orchards receiving both monocalcium arsenite and bordeaux, therefore, an average of 21 percent for the former did not seem unreasonably high. Consequently, the data pertaining to control of the disease in table 1 were computed from this average. It may be seen that the arsenite spray reduced sporodochial development by 58 to 91 percent and twig infection by 75 to 99 percent regardless of the other materials added.

The lowest reduction both in sporodochial development and in incidence of the disease occurred in test orchard No. 2. Here both the eradicant spray and the protective spray were applied with a mechanically-operated set of spray nozzles. Here, also, occurred the greatest variability in the sporodochial numbers from one part of the trees to another. These structures were much more numerous on twigs taken from peripheral branches than those taken from branches near the center of the tree. Apparently, therefore, incomplete coverage of the twigs by the eradicant spray was involved. Sporodochial numbers in test orchards 1 and 3, on the other hand, varied little from one part of the tree to another. These orchards were sprayed by hand-operated nozzles.

Judging from the data in table 1, the addition of bordeaux, 4-4-100, and 1/2 percent petroleum oil neither increased nor significantly decreased the eradicant action of a 3-100 preparation of monocalcium arsenite.

DIVISION OF PLANT PATHOLOGY OF THE UNIVERSITY OF CALIFORNIA, DAVIS, CALIFORNIA

SULFUR INJURY TO SOUR CHERRY PETALS

F. H. Lewis and R. S. Kirby

For some 10 or 12 years prior to 1948, control of the cherry leaf spot fungus, Coccomyces hiemalis Higgins, was the most important factor in cherry production in southern Pennsylvania. Following defoliation by leaf spot in midsummer of 1945, the trees bloomed unevenly in 1946, with considerable variation in the development of leaves during bloom and in the time of petal-fall. In both 1946 and 1947, it was obvious that numerous leaf spot infections occurred during bloom. In 1947, an experimental application of Compound 341 hydrochloride before bloom reduced the percentage infected leaves on May 29 to June 3 to 1.5 percent whereas adjacent trees not sprayed before bloom averaged about 25 percent infected leaves.

In line with this evidence, we have recommended sprays starting at the pink stage on sour cherry for the control of leaf spot for the last two years. Lime sulfur at 2 gallons per 100 gallons of spray was used by practically all growers in 1948. No unusual injury occurred with this or any of the materials used experimentally.

Our experience in 1949 was not so fortunate. Tree growth began from one to two weeks earlier than normal with several periods of cold weather and frost in April and early May. On April 23, when sour cherries were approaching full bloom, it was found that the petals were dead in some orchards in Adams County while others showed no injury. Wherever the injury was found, it was present whether or not the petals on individual flowers were exposed. A study of spray material comparisons and adjacent sprayed and unsprayed orchards in the area proved beyond question that the injury was caused, directly or indirectly, by sulfur applied just before bloom. Most of the injury was on trees sprayed with lime sulfur. In one instance, lime sulfur in the pink spray killed about 80 percent of the petals while adjacent unsprayed trees were not injured. Sulfur paste caused much less injury than lime sulfur. No injury was observed where Bordeaux mixture was used.

The bloom period of 1949 was unusually favorable for bee flight, and the bees seemed to work the injured flowers as well as the uninjured ones. The trees set a fair crop of fruit in spite of the petal injury.

Even though the injury occurred only where the trees were sprayed with sulfur, it is felt that the effect of the sulfur was indirect and that the actual petal killing was due to low temperature. The stage of the buds at the time the spray was applied, and the thousands of injured flowers still tightly enclosed in the bud on April 23, argue against any direct burning by the spray.

In 1950, lime sulfur will likely be replaced by Bordeaux mixture or one of the proprietary copper compounds.

PENNSYLVANIA STATE COLLEGE, STATE COLLEGE

FURTHER EVIDENCE THAT IMMUNE RIBES DO NOT INDICATE PHYSIOLOGIC RACES OF CRONARTIUM RIBICOLA IN NORTH AMERICA

Glenn Gardner Hahn

In a recent paper (3) the writer presented collected data relating to the Viking (Syn. Rød Hollandsk Drueribs, Holländische Rote) red currant and its long history of freedom from the establishment of Cronartium ribicola A. Fisch. upon its leaves. In doing so he pointed out the fact that if physiologic races of the pine parasite exist, experience with Viking based on a very extensive blister-rust exposure over many years in Europe and North America, has not indicated their presence. Boyce, in the recent second edition of his text book, makes the statement that there is no evidence so far shown that there is more than one physiologic race of Cronartium ribicola (1, p. 206). We now have the same kind of evidence as that provided by Viking in the case of the two rust-immune Canadian black currant hybrids, namely No. O-381 and O-393 (2, 5), known by the varietal names "Crusader" and "Coronet", respectively. Experience in North America with these recently discovered horticultural ribes has shown the new creations to be completely free from rust infection, despite exposure to C. ribicola under conditions of natural infection on numerous blister-rust sites both in the eastern and western parts of this continent.

In the United States the Canadian rust-immune black currant hybrids have not been tested experimentally with Cronartium ribicola from the Pacific Northwest where the parasite was introduced originally on a single shipment of infected Pinus strobus L. from France (1, pp. 206-207). Viking has been planted in the western blister-rust area, however, and has remained free from rust development (3). In the East where several introductions of rust-infected P. strobus from Europe occurred (1, p. 206), and where the widely tested Viking has been free from rust establishment for years (3), the new Canadian black currant hybrids also have shown themselves to be immune under conditions of controlled experimentation when tested with rust collected in four widely separated States, namely Connecticut, Maine, Virginia, and Wisconsin (2, 4).

In Canada, on the other hand, where Viking has been tested only in blister-rust territory at the Central Experimental Farm, Ottawa, and where since 1932 it consistently has remained free from rust development, the new rust-immune black currant hybrids have had a most comprehensive exposure to Cronartium ribicola that Viking did not experience. This extensive exposure was designed purposely by the Canadian originators of the hybrids because of the lack of formal experimentation to prove or disprove the actual existence of physiological races of the parasite. Accordingly they adopted the next best step and distributed their hybrids as widely as possible over blister-rust territory in the expectation that

should physiological races of ribicola actually exist, one or the other of their selections might not prove to be immune under conditions of natural infection.

For the purpose of this paper, Dr. A. W. S. Hunter has most generously contributed the carefully checked Canadian rust-exposure results. At the Central Experimental Farm where rust infection occurs yearly in the ribes plantations maintained there, observations on the new hybrids made by him and also by different members of the staff of the Division of Botany and Plant Pathology did not reveal rust infection. The hybrids have been cultivated continuously at Ottawa since their discovery in 1941. Similar results were obtained at the following stations where the hybrids have been tested since 1945: in eastern Canada--Prince Edward Island (Charlottetown), Nova Scotia (Kentville, Truro), Province of Quebec (Ste. Anne de la Pocatière, Ste. Anne de Bellevue (Macdonald College), L'Assomption, Normadin, Chateauguay, Quebec (Department of Agriculture)), Ontario (Harrow, St. Catherines, Hamilton, Vineland, Kemptville, Kapuskasing), New Brunswick (Fredericton); and in western Canada--British Columbia (Saanichton and Victoria on Vancouver Island, Agassiz, Abbotsford, Nelson). At the stations mentioned observations were made by the resident plant pathologist or horticulturist thoroughly familiar with blister rust.

Conclusion

Evidence for the non-existence of races of Cronartium ribicola supplied by our field observations and experimental results with the Viking red currant and the two Canadian black currant hybrid selections does not in itself indicate that rust strains definitely do not exist. The evidence does indicate, however, that if strains of the parasite do occur in North America they do not grow and fruit on these three rust-immune ribes.

Literature Cited

1. Boyce, J. S. Forest Pathology. Ed. 2. 550 pp. New York. 1948.

2. Hahn, G. G. Immunity of Canadian black currant selections from blister rust. Phytopath. 38: 453-456. 1948.

3. _____ Evidence of the non-existence of physiological races in Cronartium ribicola. Phytopath. 39: 85-87. 1949.

4. _____ and A. V. Miller. Rust immunity of Canadian black currants under field conditions in Connecticut. U.S. Bureau of Plant Industry, Soils, and Agricultural Engineering, Plant Disease Reporter 33: 275-276. 1949.

5. Hunter, A. W. S., and M. B. Davis. Breeding rust-resistant black currants. Am. Soc. Hort. Sci. Proc. 42: 467-468. 1943.

DIVISION OF FOREST PATHOLOGY, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, U. S. DEPARTMENT OF AGRICULTURE, IN COOPERATION WITH YALE UNIVERSITY, NEW HAVEN, CONNECTICUT

BACTERIAL LEAF SPOT OF PELARGONIUM IN TEXAS

E. M. Hildebrand and A. C. Yezak

A bacterial disease of a cultivated species of Pelargonium, originally recorded from the vicinity of Austin and San Antonio, Texas, by Heald and Wolf in 1912, was reported in detail by Lewis (5, 1914) and the causal organism was named Bacterium (Pseudomonas) erodii. It is now listed in Bergey's Manual (1) as Pseudomonas erodii. Specimens of a bacterial leaf disease of greenhouse-grown geraniums have been brought to our attention several times during the past three years. One florist in particular stated that much damage was being done to his plants.

In attempting to diagnose the trouble isolations were made and the organism was studied in some detail. It seems to be distinct from the bacterial leaf spots caused by Xanthomonas pelargonii and X. geranii, respectively, reported by Brown (1923) and Burkholder (1937) (2,3).

The susceptible hosts of this organism according to Lewis (5) include the following cultivated varieties of Pelargonium: American Beauty, Anna Grothe, Colonel Poirine, Dagata, Jubelpaar, Lucifer, Beauté Poitevine, Columbia, Jean Viand, John Doyle, La Favorite, Mrs. Frances Perkins, S. A. Nutt, ivy-leaved-L'Etincelant, Souvenir de Chas. Turner, Beauty of Jersey, Joan of Arc; variegated-leaved Madam Salleroi; pansy geranium Mrs. Loyal. Besides these, Mme. Buchner and Mme. Landry have been proved susceptible at College Station. The bedding plant, Pelargonium hortorum, was found insusceptible in greenhouse tests.

The wild crane's bill, Erodium texanum, a native plant to central Texas, is the only wild plant known to be susceptible to infection by this organism. The best common name is the bacterial leaf disease of Erodium and Pelargonium since susceptible species have been found only in these two genera.

The diseased specimens from which cultures have recently been isolated and studied were collected in greenhouses at Waxahachie and College Station on Pelargonium.

Symptoms of natural infections of this disease have similarities and differences on Erodium and Pelargonium.

In Erodium the newest spot infections are reddish-brown but change readily to black, becoming watery in appearance. Some of the spots are circular, but more frequently they enlarge and become triangular with the apex in the leaf interior and subtended by an expanding arc extending to the leaf margin. Affected tissue soon becomes dry, retaining its dark color or becoming brownish. As the disease progresses the lesions enlarge until nearly if not all of the leaf becomes necrotic and

On Pelargonium the symptoms were uniform on all varieties observed. Infection first became noticeable as a transparent dot. The spot gradually became larger with the center reddish-brown in color and the outer edges almost colorless. At maturity the spots were 2 to 5 mm. in diameter. The characteristic of including areas between main veins, which is present in Erodium, is also exhibited by Pelargonium species. Progression of the disease is different, being always from the margin toward the major veins and base of the leaf. This symptom pattern would suggest that the organism might be entering Pelargonium through the leaf margin openings or hydathodes.

Lewis (5) considered Erodium texanum as the primary native source of inoculum, since he found this wild plant commonly infected at Austin and San Antonio. While Erodium texanum is native (4) to all localities where bacterial leaf spot of Pelargonium has been found, the Erodium plants in the College Station locality have not been observed with the foliage symptoms. Incidentally, however, many Erodium plants have been severely affected with a downy mildew the past three spring seasons. It is possible other wild hosts may perpetuate the organism but this possibility has not been examined.

It appears on the basis of these preliminary observations that the organism described by Lewis (5) thirty-five years ago should be re-examined in the light of the present. While a preliminary re-examination of the morphology and physiology of the causal organism agrees in the main with the original description as given by Lewis, too little is known about the life history of the organism to enable us to formulate control measures.

Literature Cited:

1. Breed, R. S., et al. Bergey's Manual of Determinative Bacteriology. 6th Edition. Williams and Wilkins. Baltimore. 1948.

2. Brown, N. A. Bacterial leaf spot of geranium in the eastern United States. Jour. Agr. Res. 23: 361-372. 1923.

3. Burkholder, W. H. A bacterial leaf spot of geranium. Phytopath. 27: 554-556. 1937.

4. Cory, V. L., and H. B. Parks Catalogue of the flora of the State of Texas. Texas Agr. Exp. Sta. Bul. 550. 1937.

5. Lewis, I. M. A bacterial disease of Erodium and Pelargonium. Phytopath. 4: 221-231. 1914.

TEXAS A & M COLLEGE, COLLEGE STATION

RESULTS FROM COOPERATIVE TESTS OF CEREAL SEED TREATMENTS

R. W. Leukel

During the 1948-49 crop season uniform cooperative field experiments were carried out at several stations in seven Southern States to determine the relative effectiveness of certain fungicides for the control of several seed-borne diseases of cereals.

The following fungicides were used:

1. New Improved Ceresan, (5% ethyl mercury phosphate).

2. Ceresan M (7.7% ethyl mercury p-toluene sulfonanilide).

3. Ceresan M slurry (same as No. 2).

   Nos. 1, 2, and 3 are made by the Dupont Company and were applied at the rate of 1/2 ounce per bushel.

4. Spergon (98% tetra chloro para benzoquinone)

5. Phygon XL (50% dichloro naphthoquinone)

   Nos. 4 and 5 are made by U. S. Rubber Company and were applied at the rate of 2 ounces per bushel.

6. Arasan (50% tetramethyl thiuram disulfide)

7. Arasan S.F. (75% " " ")

   Nos. 6 and 7 are Dupont products. No. 6 was applied as a dust at 2 ounces per bushel and No. 7 as a slurry at the rate equivalent to that of No. 6.

8. Seedox (50% tri chlorophenol acetate) is made by R. J. Prentiss & Company and was applied at 2 ounces per bushel.

9. Panogen (1.2% methyl dicyandiamide) is marketed by T. M. Duche and Sons, Inc. This is a liquid fungicide and was applied at the rate of 2 fluid ounces per bushel, the grain being mixed in a treater as in the application of dusts. The product now on the market contains 2.1% methyl mercury dicyan-diamide and the recommended rate of application to small grains is 3/4 fluid ounce per bushel.

Table 1. Emergence from seed of wheat, barley and oats, untreated or treated as shown, and planted in non-sterile soil 50 percent saturated 2 or 3 weeks after treatment, and kept at 16-18°C. during emergence, Beltsville, Maryland September 1948.

Seed treatment			Percent emergence from seed of:-			
No.	Material	oz. per bu.	Wheat	Barley[a]	Barley[b]	Oats
1	N. I. Ceresan	0.5	60	89	94	97
2	Ceresan M	0.5	61	83	95	94
3	Ceresan M slurry	0.5	80	81	88	93
4	Spergon	2.0	78	92	93	91
5	Phygon XL	2.0	79	89	91	93
6	Arasan	2.0	79	93	97	93
7	Arasan S. F.	2.0	78	94	97	95
8	Seedox	2.0	37	72	86	87
9	Panogen	2.0	66	88	92	96
10	Check	-	54	87	89	90

[a] stripe-infected seed. [b] smut-infected seed.

The following lots of seed were used:

Victorgrain oats naturally infected with loose smut (Ustilago avenae) and covered smut (U. kolleri) was obtained from O. S. Bowers, Siler City, North Carolina. Seed of a mixture of barley varieties naturally infected with stripe disease (Helminthosporium gramineum) was furnished by T. T. Hebert of Raleigh, North Carolina. Seed of barley (probably a mixture of Missouri Early Beardless and Kentucky No. 1) infected with covered smut (Ustilago hordei) was supplied by D. A. Reid of Lexington, Kentucky. Purplestraw wheat, grown at Beltsville, Maryland, was inoculated with spores of bunt (Tilletia foetida) at a spore dosage of 1 to 200 by weight.

Procedure

The different seed lots were first thoroughly cleaned by fanning and screening. The treatments were applied in flat pans with tightly fitting covers and with baffles inside. The pans were first "conditioned", each with a different fungicide, by treating rather heavily seed that was subsequently discarded. Then 1000 cc. (1/35 bushel) of the seed to be planted was placed in each of the pans, the proper amount of fungicide

was added and the pans were revolved in a mechanical device for 15 minutes. The treated seed was kept in open containers until it was packeted for sowing in the field. Germination tests were made in non-sterile soil, 50 percent saturated and kept at 16-18° C. Three replicates of 100 seeds each were planted for each combination of seed and fungicide.

The seed was packeted at Beltsville for sowing in triplicated rod rows at nine stations in six States. The names of the stations and of the staff members through whose generous cooperation these experiments were carried out are as follows:

Beltsville, Maryland	J. W. Taylor
Raleigh, North Carolina	T. T. Hebert
North, South Carolina	C. H. Arndt
Clemson, South Carolina	C. H. Arndt
Richberg, South Carolina	C. H. Arndt
Experiment, Georgia	U. R. Gore
Gainesville, Florida	S. C. Litzenberger
Quincy, Florida	W. H. Chapman
Lexington, Kentucky	D. A. Reid

In taking data on disease control, the stripe-infected barley plants or the smut-infected heads of barley, oats, and wheat in each row were carefully counted. An approximation of the total number of plants or heads per row was arrived at by making actual counts in representative rows or parts of rows, and then supplementing these counts by careful estimates.

Experimental Results

The data on emergence are shown in Table 1. Seedox significantly reduced emergence in wheat and in the stripe-infected barley. Four non-mercurials and Ceresan M slurry seemed to increase emergence in wheat significantly. The untreated barley and oats germinated so well that significant increases were hardly possible.

The data on disease control are shown in Table 2. At Lexington, the oats winter-killed and at the Florida stations the barley failed to head. At Richberg only oats were sown. The four mercurials were the only materials to completely eliminate the diseases under study in the four seed lots. The failure of the non-mercurials to control stripe disease and covered smut in barley and the smuts in oats satisfactorily should eliminate them from further consideration as suitable fungicides for these cereals.

No bunt developed at four of the stations and infection was relatively light at the other four. This was due probably to warm weather following planting. The four mercurials eliminated all trace of bunt

Table 2. Relative effectiveness of different fungicides in the control of certain seed-borne diseases of cereals.

Crop and disease	Location	Percent infection in plants from seed treated with [a]										Percent from untreated seed
		N. I. Ceresan	Ceresan M	Ceresan M slurry	Spergon	Phygon	Arasan	Arasan S. F.	Seedox	Panogen		
Stripe disease in mixture of barley varieties	Beltsville, Md.	0	0	0	6.0	3.0	2.0	1.0	2.0	0		14.0
	Raleigh, N.C.	0	0	0	2.0	1.3	1.3	3.0	2.0	0		3.0
	North, S.C.	0	0	0	4.9	2.6	2.1	1.9	2.3	0		18.0
	Clemson, S.C.	0	0	0	8.1	4.6	6.0	7.7	8.0	0		11.9
	Experiment, Ga.	0	0	0	1.1	1.4	0	1.3	1.5	0		2.6
	Lexington, Ky.	0	0	0	12.3	6.0	4.3	6.3	6.7	0		17.3
	Average	0	0	0	5.7	3.2	2.6	3.5	3.8	0		11.1
Covered smut in mixture of Missouri Beardless and Kentucky No. 1 barleys	Beltsville, Md.	0	0	0	.4	.1	.1	.2	2.4	0		8.7
	Raleigh, N.C.	0	0	0	.3	.1	0	.3	1.8	0		1.9
	North, S.C.	0	0	0	0	0	0	0	1.2	0		1.8
	Clemson, S.C.	0	0	0	.8	.2	0	0	2.7	0		2.0
	Experiment, Ga.	0	0	0	.1	0	0	.1	.5	0		1.4
	Lexington, Ky.	0	0	0	1.8	0.7	1.0	1.8	11.6	0		15.3
	Average	0	0	0	.6	.2	.2	.4	3.4	0		5.2
Covered and loose smut in Victorgrain oats	Beltsville, Md.	0	0	0	7.1	4.0	6.0	8.4	3.2	0		22.4
	Raleigh, N.C.	0	0	0	5.3	3.1	8.1	4.7	3.7	0		11.6
	North, S.C.	0	0	0	4.2	.7	2.8	2.2	.6	0		9.0
	Clemson, S.C.	0	0	0	4.4	2.2	5.4	4.9	3.5	0		11.3
	Experiment, Ga.	0	0	0	6.1	3.1	6.5	6.4	2.4	0		8.0
	Richberg, S.C.	0	0	0	2.2	1.8	2.4	4.1	0.9	0		7.8
	Gainesville, Fla.	0	0	0	4.2	1.5	2.2	6.0	0.6	0		8.1
	Average	0	0	0	4.8	2.3	4.8	5.2	2.1	0		11.2
Bunt in Purplestraw	Beltsville, Md.	0	0	0	.6	.2	.8	1.2	3.0	0		19.1
	Raleigh, N.C.	0	0	0	0	0	0	0	1.0	0		9.7
	Clemson, S.C.	0	0	0	0	0	0	0	0	0		3.3
	Lexington, Ky.	0	0	0	0	0	0	0	.8	0		5.0
	Average	0	0	0	.2	.1	.2	.3	1.2	0		9.3

[a] New Improved Ceresan, Ceresan M, and Ceresan M slurry were applied at the rate of 1/2 oz. per bushel; Spergon, Phygon, Arasan, Arasan S. F., Seedox, and Panogen at 2 oz. per bushel.

where it appeared. Some of the non-mercurials seemed to show considerable merit as treatments for the control of bunt and perhaps could be used for that purpose when more effective materials are not available.

DIVISION OF CEREAL CROPS AND DISEASES, U. S. DEPARTMENT OF AGRICULTURE, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

NEW OR UNUSUAL RECORDS OF PLANT DISEASES

BARLEY STRIPE IN CALIFORNIA

By Coit A. Suneson

Barley stripe [Helminthosporium gramineum] is definitely the most serious barley disease in California this year, and is much more prevalent than in any of the past 15 years. In a random survey of 99 fields from Stanislaus County north to Glenn and Butte Counties only 50 were free from stripe, and 24 ranged from 10 to 70 percent infection. This suggests that at least one-fourth of the 1949 northern California acreage was sown without any kind of seed treatment.

One 80-acre field of Atlas barley, which averaged 70 percent stripe so heavily infected that the estimates were rechecked. Three observers, each making two counts, found a range of from 61 to 80 percent.

Spore dispersal must have been extensive in 1948 because seed from a stripe-free field of Club Mariout one-half mile from a source of infection produced 9 percent stripe in 1949, when planted without seed treatment. A total of 55 out of 60 random market lots drawn from the 1948 crop, and planted on the University Farm without seed treatment, showed a trace to 60 percent stripe.

DIVISION OF CEREAL CROPS AND DISEASE, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING

DOWNY MILDEW ON OATS IN COLORADO IN 1946

By T. R. Stanton and R. F. Eslick[1]

A collection of named oat varieties, including some submitted for identification, was sown on the Agronomy Farm of the Colorado Agricultural Experiment Station at Fort Collins, Colorado, in the spring of 1946. In two of the identification samples, labeled Colorado Black Winter and Reitmeyer Winter but later identified as Colburt and Kanota, respectively, an estimated 25 percent of the panicles were variously contorted and proliferated. Affected panicles were collected in August, 1946, and sent to Dr. A. G. Johnson of the Division of Cereal Crops and Diseases, who identified the malady as Downy Mildew (Sclerospora macrospora Sacc.). The oospores of the disease between the veins of the leaves were plainly visible with a small hand lens.

A few similar abnormal plants with proliferated panicles also were observed at the same time in Brunker, Fulghum, Victoria, and several other red oat varieties.

DIVISION OF CEREAL CROPS AND DISEASES, BUREAU OF PLANT INDUSTRY, SOILS, AND AGRICULTURAL ENGINEERING, FT. COLLINS, COLORADO

[1] Senior Agronomist, Division of Cereal Crops and Diseases, Bureau of Plant Industry, Soils, and Agricultural Engineering, Agricultural Research Administration, U. S. Dept. of Agriculture; formerly Assistant Agronomist, Agricultural Experiment Station, Ft. Collins, Colorado

PERONOSPORA SCHACHTII ON BEETS IN NEW YORK

By Charles Chupp and L. A. Alvarez-Garcia

One of the seed companies in New York State, as usual, had its beet seed grown on the West Coast during 1948. One of the growers who bought this seed complained that his greenhouse crop of beets was almost destroyed by some disease. Consequently a generous sample of the seed was sent to the authors for examination. At first it was supposed that Cercospora beticola might have been the offender. But neither the concentrated settlings after centrifuging nor microscopical examination of fresh seeds and those incubated in moist chambers showed any trace of Cercospora.

Every mount, however, showed numerous bodies that in shape, size, coloring, and markings resembled the oospores of the downy mildew, Peronospora schachtii. There has yet been no success in germinating these and until that can be done or further infection obtained in the greenhouse, the evidence is not complete that this is the fungus causing the damage. But the fact that Leach (Hilgardia 6: 203-251. 1931) reported the mildew on beet seeds in California and the fact that the disease in the greenhouse mentioned above was reported as being especially severe, lead us to the conclusion that the Peronospora oospores probably are carried in large numbers with beet seed and when conditions are favorable for the pathogen, they can do serious damage in the seed bed. It has never been reported in New York State as present in the field.

PLANT PATHOLOGY DEPARTMENT, CORNELL UNIVERSITY

SLIME MOLD ON STRAWBERRY IN TEXAS

By E. M. Hildebrand

In early May 1949 specimens of bearing strawberry plants from a neighborhood garden with a peculiar leaf condition were brought into the laboratory for diagnosis of the trouble. Something of fungus nature had grown up the plant surface involving half or more of the leaf surface from the soil line and had covered the leaves to the extent that shading was seriously affecting the vitality of the plants. The organism involved was a slime mold identified as Diachea leucopoda that was abundantly fruiting at the time of examination. This occurrence was coincident with cool, humid weather and on plants growing on a rich soil containing an abundance of organic matter.

During the same season there was an abundance of Physarum sp. slime molds fruiting on Bermuda, St. Augustine, and other grasses. Leaves shaded by the slime mold growths always faded, wilted, and fell off the plants.

Thus, although saprophytic, the slime molds produce plant injury. Is not this injury abnormal and a disease?
TEXAS A & M COLLEGE, COLLEGE STATION

† JASMINE GALL IN TEXAS †

By. E. M. Hildebrand

During the past year jasmine galls have been received from several Texas localities including Arlington, San Antonio, and Renner, being present in nursery plantings, in home gardens, and in hedges. The plants were identified as winter jasmine, Jasminum nudiflorum. Such galls occurred in all instances on the aboveground stems and ranged in size up to about one inch in diameter. Mature galls were most prevalent in the autumn and young galls in the spring. In November 1948 isolations were attempted from mature galls. Tissue plantings taken from the depths of the galls that had been surface sterilized by immersion in calcium hypochlorite were sterile, whereas the surface tissues yielded a fungus growth both by tissue planting and by dilution techniques of isolation. Abundant black pycnidia were found over much of the surface of the mature galls. The pycnidia liberated an abundance of spores when mature galls were immersed in water. Healthy jasmine and privet plants when atomized with suspension produced galls only when the stems and petioles had been previously wounded by needle punctures. The galls formed at the wounds and required about five months to reach maturity.

The causal organism may be the so-called Phomopsis fungus which Brown (1936)[1] reported as causing privet and jasmine galls in the District of Columbia.

Further details will be reported elsewhere.
TEXAS A & M COLLEGE, COLLEGE STATION

† SOME NEW REPORTS FROM ARIZONA

By J. G. Brown and Alice M. Boyle

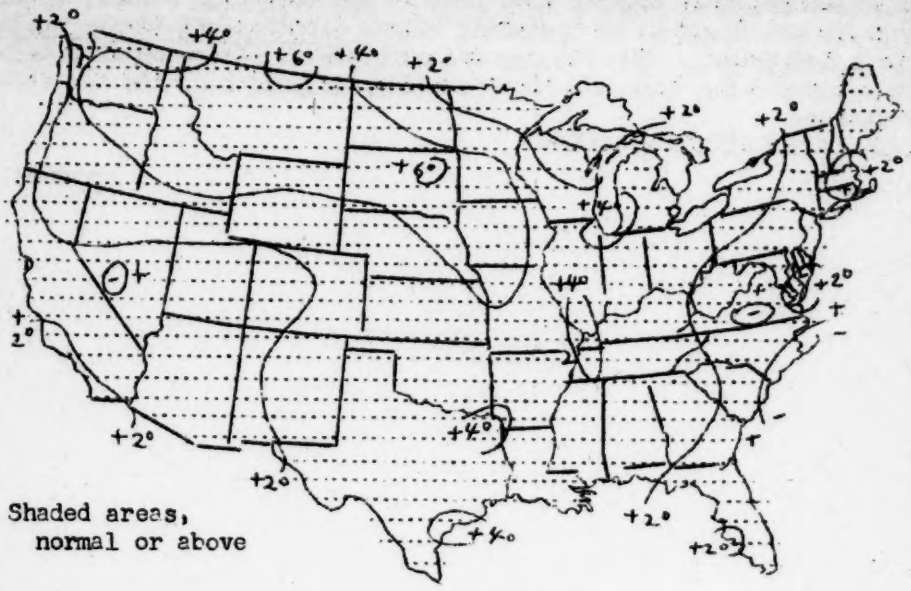
Root constriction, a physiological disease of cotton, (Hubbard, Jour. Agr. Res. 44: 39-47. 1932) was reported June 13 from Casa Grande. The trouble has previously been reported from California and Texas, but this is believed to be the first report in Arizona.

The browning and stem-break organism, Polyspora lini Laff., was isolated from specimens sent in from Deer Valley and Safford. This is the first report of occurrence in Arizona.

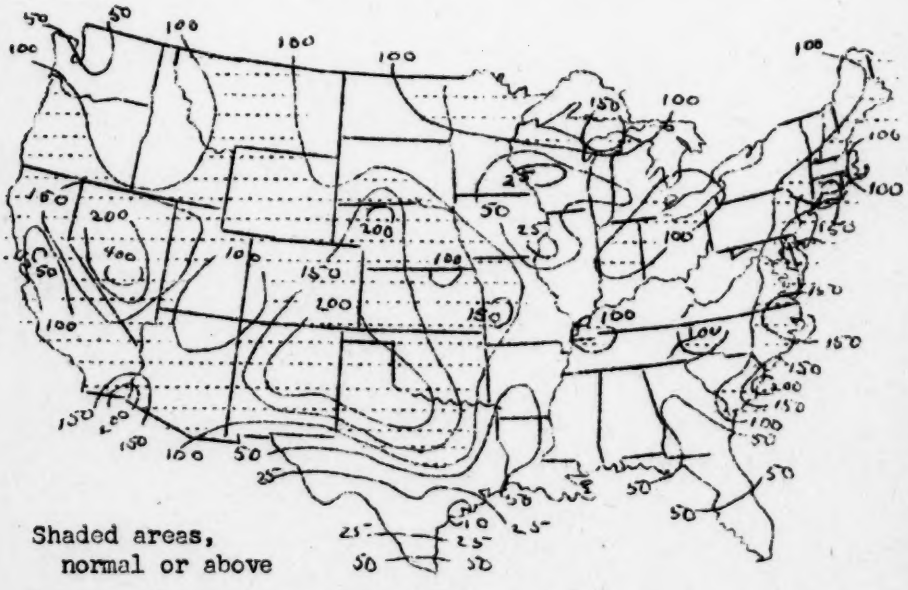
[1] Brown, N. A. Privet and jasmine galls produced by a species of Phomopsis. Phytopath. 26: 795-799. 1936.

Diseased oriental poppies were sent in for diagnosis from Flagstaff. The cause was found to be bacterial blight (Xanthomonas papavericola Bryan & McWhorter). The disease was reported to be common in poppies in flower gardens but this was the first identification of the bacterium in the State.

UNIVERSITY OF ARIZONA, TUCSON



Map I. Departure of mean temperature from normal for May, 1949



Map II. Percentage of normal precipitation for May, 1949

MAY WEATHER

(From U.S. Department of Commerce, Weather Bureau, Weekly Weather and Crop Bulletin for the week ending June 7, 1949